

Treatment of Cardiac Arrhythmias

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. David W. Martin, Jr., Professor of Medicine, and James L. Naughton, Assistant Professor of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine. Requests for reprints should be sent to the Department of Medicine, University of California, San Francisco, San Francisco, CA 94143.

DR. SMITH:* *In this conference five participants will discuss new and innovative approaches to the management of cardiac arrhythmias. The presentation will be started by Dr. Katzung.*

Basic Mechanisms of Cardiac Arrhythmias

DR. KATZUNG:† Our understanding of the basic cellular electrophysiology of cardiac arrhythmias is based largely on information provided by studies using microelectrodes.^{1,2} This technique permits the recording of the transmembrane potential of individual cardiac cells and therefore, by extrapolation, indicates the relationship of cellular activity to the surface electrocardiogram.

An important result of the earliest applications of this technique¹ was the discovery that the various types of cells in the heart normally manifest rather different transmembrane action potential characteristics. In fact, each of the major tissue types within the heart—sinus node, atrial muscle, atrioventricular node, Purkinje fibers, and ventricular muscle—has its own characteristic pattern (Figure 1). The normal cardiac rhythm is the

result of the integration of normal *automaticity* of the pacemaker cells of the heart—in the sinus node—with normal *conduction* of the impulse by the other cardiac cells. Therefore, normal sinus rhythm implies normal automaticity and conduction; conversely, any arrhythmia involves some abnormality of automaticity, of conduction, or of both.

A typical normal transmembrane action potential is shown schematically in Figure 2A. Fully polarized cells have a rapid upstroke (phase 0) and conduct impulses at high velocity (0.5 to 3 m per second); that is, they have “fast responses.” When cardiac cells are depolarized to levels that inactivate the sodium mechanism—positive to -60 mV—they may manifest “slow-response” potentials (Figure 2B). These slow responses have been well documented in a variety of cardiac cell types²⁻⁴ and may, in fact, represent the normal mode of activity in sinus and atrioventricular nodal cells.

Between the two extremes of normal, fast responses and slow responses, it is possible to show “depressed fast responses”; that is, action potentials that are primarily dependent on sodium channels but are sufficiently depolarized to substantially

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inactivate them.⁵ The resulting conduction velocity is between 0.05 and 0.2 m per second. The characteristics of these three classes of action

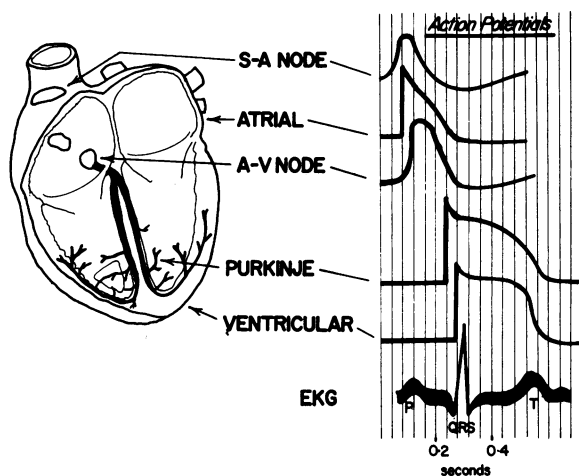


Figure 1.—Schematic diagram of various electrically important components of the heart and their typical transmembrane action potentials. Cell types that normally function as pacemakers, especially sinoatrial nodal cells, are distinguished by their diastolic depolarization and a very slow action potential upstroke. Cells in which rapid conduction is important, such as Purkinje fibers, are notable for their extremely sharp rapid upstrokes. The algebraic sum of these potentials in the extracellular space, vectored out to the body surface, yields the familiar electrocardiographic (EKG) pattern shown in the bottom tracing.

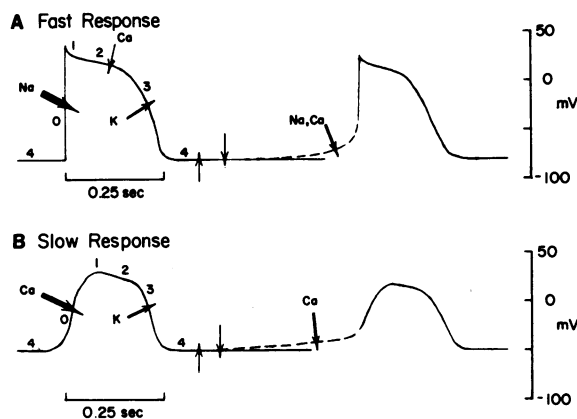


Figure 2.—A, Normal action potential as recorded from a ventricular fiber. During phase 0, a massive influx of sodium (Na) ions causes rapid depolarization from the resting potential of -80 to -90 mV to the overshoot level of $+25$ to $+30$ mV. A long plateau follows (phases 1 and 2) during which the membrane is kept depolarized by a small influx of calcium (Ca) ions. Repolarization (phase 3) is finally brought about by the increase of an outward potassium (K) current. During the ensuing phase 4 (electrical diastole), membrane potential remains constant if inward and outward cation fluxes (primarily sodium and potassium) are equal. However, if the inward depolarizing current exceeds the outward one, gradual diastolic depolarization (dashed line) takes place that may eventually reach the threshold (second action potential in upper tracing). Such diastolic depolarization is characteristic of automatic, or pacemaker, cells. B, "Slow-response" action potentials in a depolarized Purkinje or ventricular fiber. Because depolarization inactivates the sodium channels upon which the fast normal action potential depends, these depolarized cells are dependent upon the much smaller slower depolarizing effects of calcium influx. Pacemaker activity can also occur in slow-response cells.

potential responses are summarized in Table 1.

Application of these observations to the analysis of abnormal automaticity as a cause of ventricular arrhythmias shows that several types of abnormal automaticity can be defined. Automaticity induced by depolarization, such as by *currents of injury*, has been noted by a number of investigators in many types of cardiac tissue⁶ including those normally thought of as "quiescent" (for example, ventricular cells). Such automaticity usually occurs at membrane potentials characteristic of slow responses. Automaticity that occurs as a result of *metabolic* abnormalities may represent a special form of depolarization—induced automaticity. This form has clearly been shown to occur in canine Purkinje fibers exposed to high levels of carbon dioxide such as might occur after a coronary occlusion.²

Triggered automaticity has been documented recently in a number of cardiac preparations *in vitro*.⁷ It manifests typical phase 4 depolarization like other pacemaker activity but has the unusual property of being elicited by one or several properly timed extrasystoles. It can be aborted in a similar fashion. As a result, such automaticity in the *in situ* heart could masquerade as a reentry arrhythmia because starting and stopping a tachycardia by means of properly timed stimuli have been the major criteria for the clinical diagnosis of reentry mechanism arrhythmias. A related phenomenon is that of *transient depolarizations* that appear after application of positive inotropic agents such as digitalis or epinephrine. These small depolarizations (5 to 10 mV) follow repolarization of the earlier action potential and are facilitated by increasing the preceding drive rate and extracellular calcium. With sufficient facilitation, they may reach the threshold, setting off a burst of rapid repetitive action potentials that closely resemble those of triggered automaticity.⁵

The action of antiarrhythmic drugs on automaticity appears to result from suppression of inward currents during phase 4 and a reduction in diastolic depolarization (Figure 2). The more traditional drugs like quinidine and lidocaine suppress primarily the automaticity that involves sodium influx during diastole and that arise from more negative resting potentials. Drugs like verapamil suppress automaticity in slow-response cells by blocking calcium channels.³

A convincing mass of evidence indicates that ventricular arrhythmias occurring in the immediate postinfarction period are usually due to re-

entry mechanisms.⁸ Abnormal (slowed) conduction is of particular importance in reentry arrhythmias because conduction is involved in both of the prime requirements for reentry: unidirectional block and a conduction time greater than the effective refractory period of the cells in the circuit. These requirements are met in a variety of clinical conditions,^{5,8} especially those involving anatomic abnormalities of the conducting system or ischemic heart disease. Reentry does not require anatomic obstacles, special initiating stimuli, such as extrasystoles, or very long path lengths.

Actions of Antiarrhythmic Drugs

The action of antiarrhythmic drugs on reentry mechanisms is summarized in Table 2. Most of the traditional drugs and many of the experimental ones act on sodium channels as local anesthetics; that is, they decrease conduction velocity. Thus, both quinidine and lidocaine (for instance) have the same general depressant action on the cardiac membrane. However, during each cardiac cycle, sodium channels pass through three different states—rested, activated and inactivated—before returning to the rested state. The very real distinctions between quinidine and lidocaine (and others in this class) derive from important differences in the affinities of each agent for these various channel states.⁹

Verapamil is not very potent in blocking sodium channels but substantially affects calcium channels. The resulting reduction in calcium influx is responsible for the suppressive effects of this interesting new drug³ on slow responses. Propranolol may also exert a part of its antiarrhythmic

effects via a decrease in calcium-dependent slow-response activity. This concept is based on the fact that calcium channels are very sensitive to beta adrenergic stimuli and supported by the observation that important antiarrhythmic effects can be noted for propranolol at concentrations with beta-blocking effects that are too low to have local anesthetic effects.

Finally, reentry suppression via an action on potassium channels must be considered. Amiodarone, an experimental agent, substantially prolongs cardiac action potentials and effective refractory period with little effect on conduction velocity,³ an action best explained by potassium channel blockade. Quinidine, which also prolongs action potential duration and QT interval,¹⁰ may exert a part of its antiarrhythmic action through this mechanism.

Mechanisms of Supraventricular Arrhythmias in Humans

Atrioventricular Node Reentry

DR. GONZALEZ: * Although evidence has accumulated to suggest that various supraventricular arrhythmias may be related to altered automaticity¹¹ or triggered activity,¹² the most common mechanism in patients with recurrent supraventricular tachyarrhythmias appears to be reentry.¹³ Furthermore, reentry may involve "normal" cardiac conduction fibers as well as accessory pathways. The most common cause of recurrent paroxysmal tachycardia (PAT), for example, appears to be atrioventricular node reentry.¹⁴ A proposed electric model of the atrioventricular node contains

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TABLE 1.—Types of Cardiac Action Potentials

Type of Potential	Resting Potential (mV)	Conduction Velocity (m/sec)	Major Ion	Blocked By
Normal fast	—100 to —75	0.2 to 3	Sodium	Quinidine (lidocaine)
Depressed fast	—76 to —60	0.05 to 0.2	Sodium	Lidocaine (quinidine)
Slow	—65 to —40	0.01 to 0.05	Calcium	Verapamil

TABLE 2.—Action of Antiarrhythmic Drugs on Reentry Mechanisms

Action	Site	Result	Examples of Antiarrhythmic Drugs
Decrease conduction velocity . .	Sodium channels	Convert unidirectional to bidirectional conduction block	Quinidine, procainamide, lidocaine, phenytoin, disopyramide, aprindine, tocainide, mexiletine
Decrease conduction velocity . .	Calcium channels	Convert unidirectional to bidirectional block	Verapamil, propranolol
Increase refractory period	Potassium channels	Refractory period becomes longer than conduction time	Amiodarone, quinidine

two longitudinally dissociated pathways (alpha and beta) in the upper portion that funnel impulses into a final common pathway¹⁵ (Figure 3). If these pathways have differing conduction velocities and refractory periods, the proper milieu for reentry is present. A premature impulse may, for example, be blocked in one pathway but conduct via the unblocked pathway. If conduction of this premature impulse through the node is sufficiently slow, the emerging wave front may traverse the previously blocked pathway and return to the chamber of origin. Figure 3 depicts these events for a premature atrial depolarization.

A reentrant arrhythmia will be sustained providing conduction velocity in this circuit is sufficiently slow so that the emerging wave front always meets excitable tissue.

Recently, techniques have become available to detect the site and mechanism of these reentrant arrhythmias more accurately. The techniques involve insertion of multipolar electrode catheters to record atrial and His bundle deflections and cardiac pacing. In addition, the cardiac cycle may be scanned with progressively premature depolarizations to initiate and terminate tachycardias. These techniques are illustrated in Figure 4. A

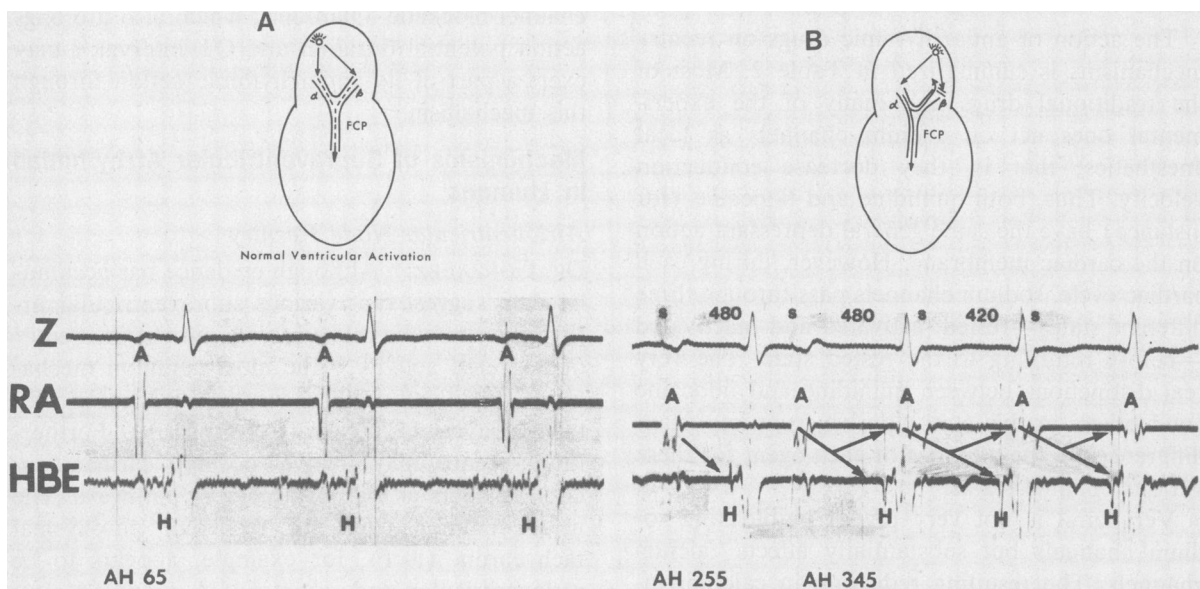


Figure 3.—Simultaneous recordings of the Z lead of the Frank orthogonal lead system and right atrial (RA) and His bundle electrograms (HBE) during sinus rhythm (A) and during induced atrioventricular nodal reentry (B). Depicted in the upper panels are electrical models of the atrioventricular node. Alpha and beta denote longitudinally dissociated pathways in the upper portion of the node that funnel impulses into a final common pathway (FCP). During sinus rhythm, the atrioventricular nodal conduction time is 65 msec. The right lower panel shows the atrium drive at a cycle length of 480 msec (S-S=480) that results in atrioventricular nodal Wenckebach conduction. The second atrial complex results in a reentrant supraventricular tachycardia whose circuit is defined by the arrows. During tachycardia, the atrial deflection coincides with QRS, thereby excluding participation of an accessory pathway. The last two stimuli occur while the atrium is still refractory.

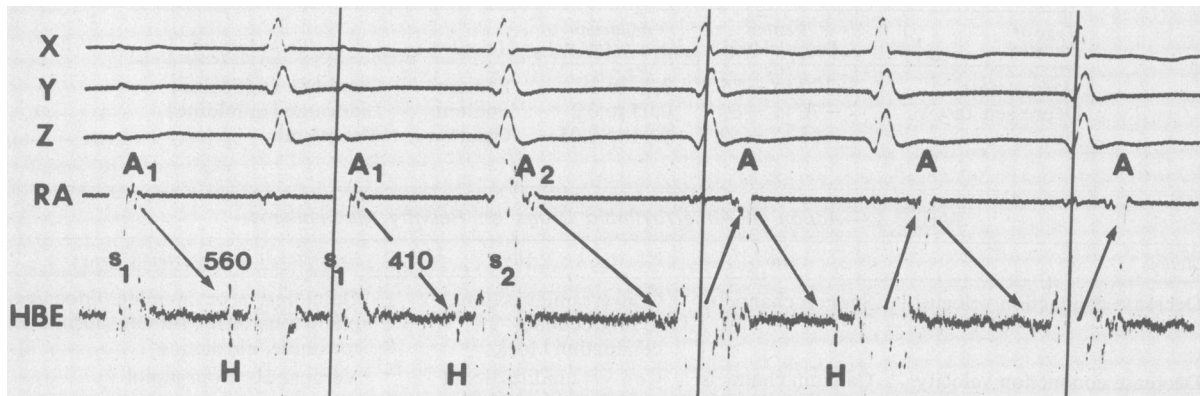


Figure 4.—Simultaneous recording of X, Y and Z leads of the Frank orthogonal lead system, right atrial (RA) and His bundle electrograms (HBE). The atrium is being paced at a cycle length of 560 msec (S-S). An atrial premature beat induced at 410 msec (S₂) following the last driven atrial complex results in prolongation of the PR interval (presumably AH) and initiates a bout of supraventricular tachycardia.

critically timed premature atrial depolarization results in a crucial delay of atrioventricular nodal conduction to allow for initiation of a sustained tachycardia.

Sinoatrial or Atrial Reentry

Reentrant supraventricular arrhythmias may also occur within the atrium or involve the sinus node.¹⁶ The basic mechanism is similar to that just described in that a premature impulse may be blocked in a portion of atrial muscle or at one margin of the sinus node and allow for initiation of a reentrant arrhythmia. Reentrant atrial or sinoatrial tachyarrhythmias commonly occur in patients with sinus node dysfunction (bradycardia-tachycardia syndrome).

Supraventricular Arrhythmias in Patients With Accessory Pathways

DR. SCHEINMAN:* Reentrant supraventricular arrhythmias may also involve an accessory pathway linking the atrium and ventricle. These pathways are known as Kent bundles and are usually involved in the tachycardia circuit. Tachycardia may develop because of differences in refractoriness between the normal (atrioventricular node-His axis) and the accessory pathway. The usual sequence of events is for a premature depolarization to block in one pathway, usually the accessory pathway, but conduct via the normal pathway and return to the chamber of origin by exciting the previously blocked pathway.¹⁷ Possible mechanisms of supraventricular tachycardia are shown in Figure 5. Patients with the Wolff-Parkinson-White syndrome are at risk of developing two types of supraventricular arrhythmias: reentrant supraventricular tachycardia as described earlier, which is the most common type, and atrial fibrillation with rapid conduction from the atrium or ventricle via the accessory pathway. If the refractory period of the accessory pathway is sufficiently short, life-threatening ventricular arrhythmias may develop in the course of atrial fibrillation. An illustration of rapid ventricular response in a patient with Wolff-Parkinson-White syndrome and atrial fibrillation is shown in Figure 6.

Treatment of Supraventricular Arrhythmias

Supraventricular arrhythmias resulting from sinoatrial or intra-atrial reentry are best managed

with classic type I antiarrhythmic drugs (quinidine, procainamide, or disopyramide). In addition, digitalis (or propranolol) may be required to impede conduction through the atrioventricular node. It should be emphasized that these arrhythmias may occur in patients with sinus node dysfunction, and the antiarrhythmic drugs alluded to may further suppress sinus activity. In the latter situation, consideration must be given to insertion of a permanent cardiac pacemaker to enable optimal use of the required drugs.

In patients with intranodal reentry, initial emergency treatment involves use of vagotonic maneuvers or drugs.¹⁸ Vagal maneuvers (carotid

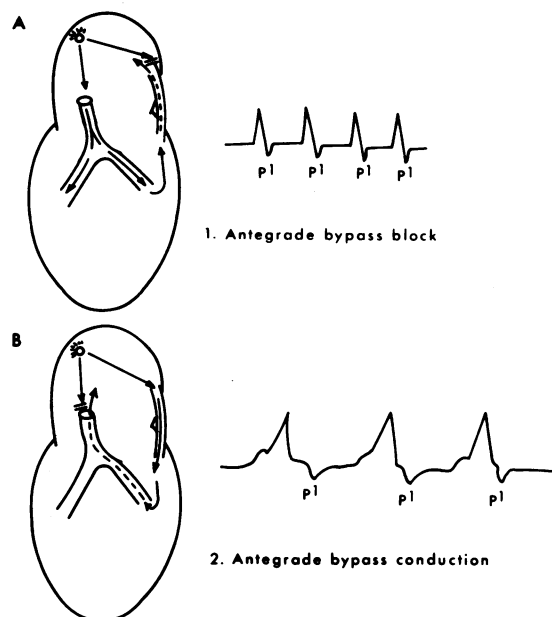


Figure 5.—In patients with the Wolff-Parkinson-White syndrome and paroxysmal supraventricular tachycardia, the reentrant circuit is usually maintained by antegrade conduction down the atrioventricular node-His bundle axis and retroconduction via the Kent bundle. **A**, The electrocardiogram will show normal QRS. **B**, Rarely there will be antegrade conduction via the accessory pathway and retroconduction via the atrioventricular node-His axis. (Reprinted with permission from Scheinman.¹⁹)

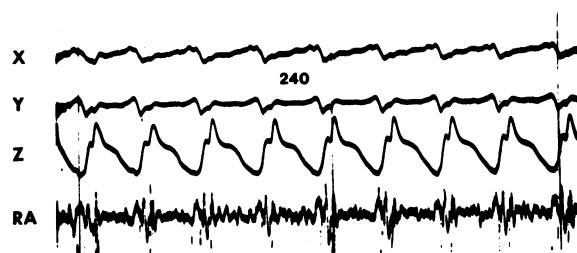


Figure 6.—Atrial fibrillation with ventricular preexcitation in a patient with the Wolff-Parkinson-White syndrome. The shortest pre-excited RR interval during atrial fibrillation was 240 msec. (Abbreviations as in Figure 3.)

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sinus pressure, Valsalva's maneuver) act to impede conduction and prolong refractoriness in the atrioventricular node, and may, therefore, extinguish intranodal reentrant arrhythmia. Similarly, digitalis, propranolol, or both, have similar effects on the atrioventricular node and have also been found to be effective in arrhythmia termination. Verapamil is an experimental drug that acts to block transmembrane calcium flux and, hence, has predominant effects on the slow-response cells. In clinical usage this drug has variable effects on sinus rate but induces substantial increases in atrioventricular nodal conduction time; it is presently the drug of choice in Europe for treatment of paroxysmal supraventricular tachycardia. The classic type I drugs appear to be less useful for acute arrhythmia control but may be effective for chronic prophylaxis. These agents may be effective either because of suppression of the premature beat that initiates the tachycardia or because of their influence on prolonging intra-atrial refractoriness so that return impulses from the atrioventricular node find an inexcitable atrium.

Drug Therapy for Arrhythmias in Patients With the Wolff-Parkinson-White Syndrome

The acute management of patients with the Wolff-Parkinson-White syndrome and paroxysmal supraventricular tachycardia is similar to that for patients with intranodal reentry. One important exception is avoidance of digitalis because this drug may shorten the refractory period of the accessory pathway.¹⁹ If atrial fibrillation should develop subsequently, ventricular fibrillation may also occur because of an extremely rapid ventricular response.²⁰ Patients with the Wolff-Parkinson-White syndrome who present with atrial fibrillation and extremely rapid ventricular response should be treated with emergency direct current cardioversion. After reversion to sinus rhythm, the patient should receive intravenous procainamide because this drug may be administered safely intravenously and it usually results in prompt prolongation of the refractory period of the accessory pathway. Chronic oral therapy with one of the type I drugs should be initiated, but the patient should undergo electrophysiologic studies including induction of atrial fibrillation, to be certain that the drug is, in fact, effective in controlling the ventricular response during atrial fibrillation (Figure 7). If a drug or drug combination cannot be found to control the ventricular

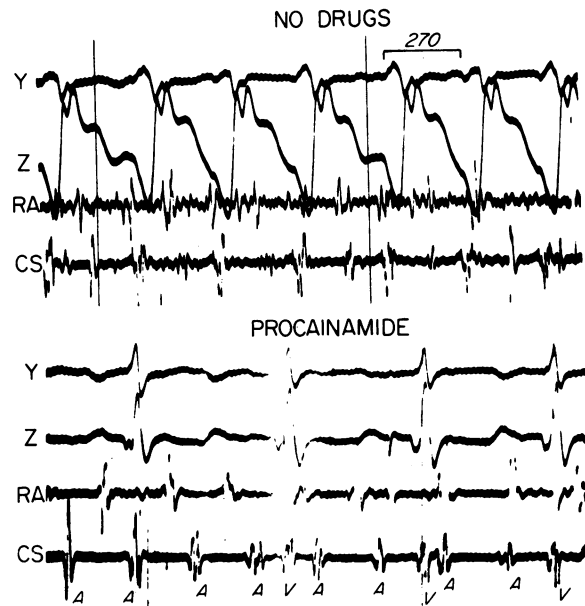


Figure 7.—The upper panel shows atrial fibrillation with rapid ventricular response; all QRS deflections show maximal preexcitation. Thus, there is a rapid rate due to antegrade conduction down the accessory pathway. In the lower panel, atrial flutter induced after intravenous infusion of procainamide shows 2:1 atrioventricular block. The QRS complexes are now conducted down the normal atrioventricular node-His axis (narrow QRS complexes) and there is complete block in the accessory pathway. (RA=right atrium; CS=coronary sinus.)

response to atrial fibrillation adequately, surgical extirpation of the accessory pathway is indicated (see subsequent section).

Management of Patients With Supraventricular Arrhythmias Refractory to Drug Therapy

If a patient has recurrent bouts of paroxysmal supraventricular tachycardia or atrial fibrillation and flutter that are unresponsive to medical management, electrophysiologic studies are indicated to define the mechanisms and tachycardia circuit, to assess the possible benefits of radiofrequency pacemaker insertion, or to determine the type of surgical approach required. In patients with paroxysmal supraventricular tachycardia, atrial overdrive pacing may be effective in terminating the tachycardia by either overdrive suppression of a focal tachycardia or, more likely, by rendering the atrium refractory to return impulses (from either the atrioventricular node or an accessory pathway). This method involves insertion of electrodes into the atrium either via a vein or by direct implantation at the time of thoracotomy. The electrodes are connected to a subcutaneously implanted radiofrequency receiver with the patient carrying a transmitter. When the tachycardia is

perceived, the transmitter is activated by the patient and electric impulses are delivered to the atrium at rates determined during electrophysiologic evaluation (Figure 8). Our experience with this technique has been detailed previously.²¹ In brief, of eight patients treated with radiofrequency pacing, one with the Wolff-Parkinson-White syndrome subsequently required a surgical operation because of recurrent atrial flutter with rapid ventricular response; the remainder are well controlled by either the pacemaker alone or with concurrent drug therapy to decrease the number of episodes of tachycardia.

Surgical Procedures for Patients With Supraventricular Tachycardia

DR. THOMAS:* Patients who are refractory to drug or pacemaker therapy should be considered for cardiac electrosurgical procedures. Such operations should never be undertaken without previous careful electrophysiologic studies to define precisely the tachycardia circuit. In our experience over the past three years, eight patients with recurrent supraventricular tachycardia without evidence of an accessory atrioventricular nodal pathway underwent His bundle section and insertion of a permanent ventricular pacemaker. The His bundle was located by means of a hand-held bipolar electrode probe, which was used to explore the area between the coronary sinus and septal leaflet of the tricuspid valve (Figure 9). The incision was made through the area showing the maximal His bundle deflection. One patient died three days after the surgical procedure following a massive cerebrovascular event, and in one patient atrioventricular conduction returned approximately six weeks after the operation. In this latter patient, supraventricular tachycardia is currently well controlled with drug therapy. The remaining six patients are asymptomatic, have returned to normal activities, and are taking no cardiac medications.

Five patients with the Wolff-Parkinson-White syndrome underwent cardiac electrosurgery in an effort to control drug-resistant recurrent supraventricular arrhythmias. One patient had a right anterior accessory pathway, one had a posterior septal pathway and three had left free-wall pathways. One of these patients with severe cardiomyopathy and mitral regurgitation died three weeks after the surgical procedure, three patients are

asymptomatic without drug therapy, and one patient had a return of accessory pathway conduction although arrhythmias are currently responsive to drug therapy.

Ventricular Tachycardia

There appears to be an increased incidence of cases of recurrent ventricular tachycardia. This arrhythmia occurs often in patients with ischemic heart disease. The standard therapeutic approach involves empiric use of available antiarrhythmic drugs with continuous monitoring of the patient's rhythm during both normal activity and exercise stress testing.²² Two major limitations of this technique are the length of time required to obtain an optimal treatment regimen and the fact that some patients have few or no premature ventricular depolarizations during tachycardia-free periods.

An important innovation in the management

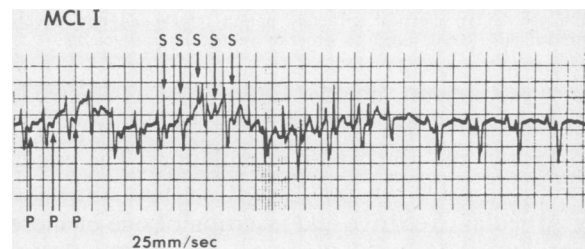


Figure 8.—Surface lead MCL I shows supraventricular tachycardia at a rate of 250 beats per minute. The inverted P waves are labeled during the tachycardia. Activation of the radiofrequency pacemaker(s) results in atrial capture at a rate of 290 beats per minute and tachycardia termination. (Reprinted with permission from Peters et al.²¹)

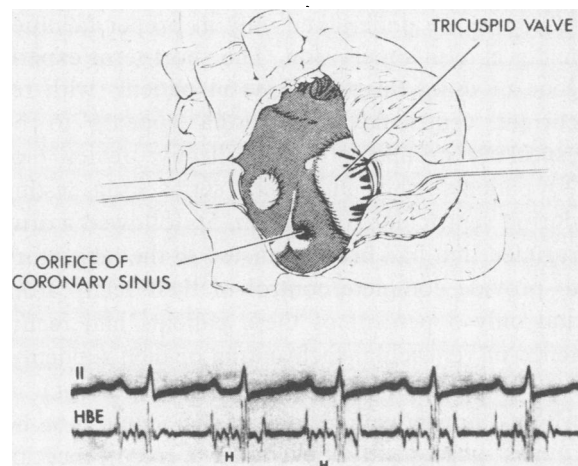


Figure 9.—The upper panel details the surgical landmarks used in identification of the His bundle. A hand-held bipolar electrode probe is used to explore the area between the orifice of the coronary sinus and the septal leaflet of the tricuspid valve. An illustration of a His bundle deflection (H) obtained during endocardial mapping is shown in the bottom panel.

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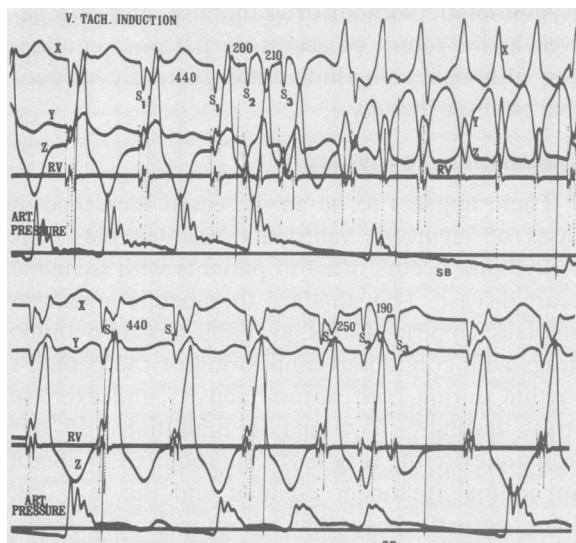


Figure 10.—**Top,** Simultaneous recordings of X, Y and Z leads of the Frank orthogonal lead system together with the right ventricular (RV) electrogram and arterial (ART) pressure curve. Sustained ventricular tachycardia was induced by insertion of two ventricular extrastimuli (S₂S₃) 200 and 210 msec, respectively, after the last driven complex (S₁). **Bottom,** After administration of procainamide, the ventricular effective refractory period lengthened and double extrastimuli (S₂S₃) failed to provoke ventricular tachycardia.

of these patients includes insertion of an electrode catheter into the apex of the right ventricle for purposes of ventricular stimulation. The study protocol calls for both short periods of rapid ventricular overdrive and insertion of one or more programmed ventricular extrastimuli throughout the ventricular diastolic cycle in an effort to provoke ventricular tachycardia.²³ The patient then undergoes serial electropharmacologic testing to determine which drug(s) prevents induction of tachycardia (Figure 10). These studies should, of course, be undertaken only in proper facilities under careful supervision. The short-term experience of using this technique in patients with recurrent ventricular tachycardia appears to be especially promising. In a collective review²⁴ of 90 patients who underwent serial drug testing during ventricular stimulation, 56 followed a drug regimen that had been predicted in the laboratory to provide complete control of their tachycardia and only 5 percent of these patients had recurrence of ventricular tachycardia or died suddenly. In contrast, 15 of 20 patients (75 percent) in whom an adequate drug regimen could not be found subsequently developed a recurrence of ventricular tachycardia or died suddenly. The benefits of the ventricular stimulation approach allow for relatively rapid detection of a drug regimen predictive of successful arrhythmia con-

trol. The risks include induction of ventricular tachycardia or ventricular fibrillation; therefore, the procedure must be done by personnel expert in cardiopulmonary resuscitation.

Surgical Management of Ventricular Tachycardia in Patients With Coronary Artery Disease

DR. ULLYOT:* The standard approach to the management of patients with recurrent ventricular tachycardia and coronary artery disease includes coronary revascularization, aneurysmectomy or both. Although the current literature contains many reports²⁵⁻³⁰ dealing with the standard surgical approach, many questions remain unanswered. Most studies tend to emphasize successful results. Most are either case reports or summaries of small series that fail to delineate carefully the preoperative medical regimen with respect to drugs used, measurement of blood levels of each drug and patient compliance. Moreover, results are reported in terms of survival statistics while the findings of continuous electrocardiographic monitoring relative to subsequent incidence of tachyarrhythmias are not given. Nonetheless, the data show an overall operative mortality of 18 percent with good results in terms of arrhythmia management in many patients.

A recent study by Mason and colleagues³¹ is perhaps the best source of information relative to this question. They report the results of 57 surgical procedures carried out over seven years for the primary indication of ventricular tachyarrhythmia in 56 patients with ischemic heart disease. Preoperative evaluation included hemodynamic studies, coronary arteriography, and left ventriculography with documentation of the recurrent nature of the arrhythmias and refractoriness to drug management. A majority of patients (58 percent) underwent combined left ventricular aneurysmectomy and coronary artery bypass grafting. Another 28 percent of the patients had aneurysmectomy alone and 14 percent had coronary artery bypass grafting alone. There were 11 intrahospital deaths or a 20 percent operative mortality, and seven of the 11 deaths were due to arrhythmias. In addition, 10 deaths occurred late in the follow-up period or at an average of 22 months. Only one of the late deaths was sudden and, therefore, presumably due to an arrhythmia. Of the 35 survivors, only 11 percent (4

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of 35) had symptomatic tachyarrhythmia. Antiarrhythmic drug therapy was required in 57 percent (20 of 35 patients). Fifteen patients had ambulatory Holter monitoring studies and four showed bouts of ventricular tachycardia. This important study shows that standard surgical techniques can be applied successfully to patients with ischemic heart disease and refractory ventricular arrhythmia with good results in many patients and with an operative mortality of approximately 20 percent. Most deaths occurred in patients with recent acute myocardial infarction or poor left ventricular function.

In an attempt to improve the standard approaches, three additional surgical procedures have evolved. The first is determining the location of the tachycardia focus by epicardial mapping following the induction of ventricular tachycardia during the operation and then carrying out a ventriculotomy over the area of earliest activation. However, this approach met with limited success.

The same group then developed a second technique; namely, "encircling myotomy."³² The technique consists of opening the left ventricle through a scarred segment and making a nearly full-thickness incision in the left ventricular endocardium at the edge of the visible endocardial scar. The incision is made perpendicular to the plane of the endocardium and is carried circumferentially around the junction of the aneurysm and normal left ventricular muscle at the edge of the endocardial scar. The incision is extended near to but not through the epicardium and care is taken not to injure the coronary arteries. The incision is closed using a continuous suture from within the left ventricular cavity.

The rationale for this procedure is to create a barrier between the aneurysm or scar and the normal muscle, and stems from recent epicardial mapping studies which showed that the origin of the tachycardia is almost always located at the border of the aneurysm. The encircling myotomy, then, provides an effective barrier that prevents reentrant ventricular arrhythmia. It should be emphasized that this border zone is usually not excised with conventional aneurysmectomy as the standard technique leaves a rim of scar tissue to ensure a secure closure. The arrhythmic focus is often located in the ventricular muscle within 2 cm of the aneurysmal scar. This may explain why the conventional approach often fails to result in tachycardia control. The originators of this tech-

nique reported on five cases of drug-resistant ventricular tachycardia that were treated with encircling myotomy.³² There were no operative deaths among the patients and drug therapy was discontinued postoperatively. No ventricular tachycardias recurred during a follow-up period of 6 to 24 months; these studies included ambulatory Holter monitoring.

The third surgical approach to treatment of recurrent ventricular tachycardia with coronary artery disease is that of endocardial excision.³³ This technique involves the use of intraoperative epicardial and endocardial mapping during induced ventricular tachycardia, paying particular attention to mapping the edges of the aneurysm. Conventional aneurysmectomy is then done and programmed ventricular tachycardia repeated. On the basis of the data obtained during tachycardia, endocardial excision is carried out in the area determined to be the origin of the arrhythmia. Those patients with substantial coronary obstructive lesions and distal vessels suitable for bypass then undergo coronary artery bypass operation. The investigators reported on 12 patients treated with this technique; there was one operative death. Postoperatively, all patients had repeated electrophysiologic studies after antiarrhythmic agents had been discontinued and in none could ventricular tachycardia be initiated. Two interesting additional observations were made in the study. In 11 of the 12 patients, tachycardia could still be induced intraoperatively after standard aneurysmectomy. This observation tends to support the notion that conventional aneurysmectomy may not be effective for arrhythmia control. One patient did not have an aneurysm, and the arrhythmic focus was detected in the posterior third of the intraventricular septum, which was excised through a ventriculotomy into normal muscle.

We have developed a surgical approach that is somewhat different from the other methods. Patients with recurrent ventricular tachycardia refractory to drug management are studied preoperatively with programmed ventricular stimulation, and after ventricular tachycardia is induced, endocardial mapping is carried out to locate the earliest areas of depolarization during tachycardia.³³ Coronary arteriography and left ventriculography are then done before electrophysiologic studies are carried out.

Coronary artery bypass grafting is done initially using moderate hypothermia (32°C) with intermittent aortic occlusion during construction of

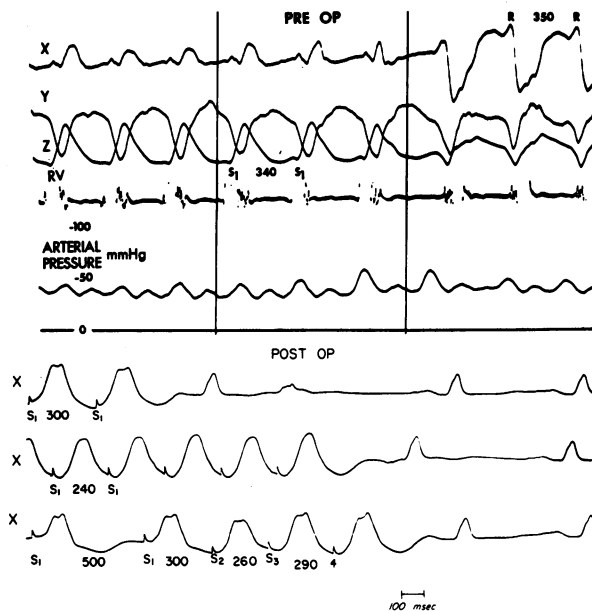


Figure 11.—**Top**, Preoperative (PRE OP) findings in a patient with recurrent ventricular tachycardia (VT) (abbreviations as in Figure 10). VT is induced after ventricular pacing at a cycle length of 340 msec. The tachycardia cycle length = 350 msec. **Bottom**, Postoperatively (POST OP), ventricular tachycardia could not be induced either with rapid ventricular overdrive pacing (CL=300, 240 msec) or by insertion of multiple ventricular extrastimuli.

a distal anastomosis. The reason for doing a coronary artery bypass first is based on two considerations. (1) Our studies of myocardium metabolism using coronary sinus catheterization showed that ischemia commonly occurs during standard coronary artery bypass operation and we believe that subsequent intraoperative electrophysiologic studies are safer when revascularization is carried out first. (2) We speculate that the augmented myocardial blood flow may change electrophysiologic events; therefore, we prefer to study patients in a state more nearly approximating their postoperative condition. In this way, we are able to assess tachycardia inducibility after coronary revascularization. In patients with either an aneurysm or large areas of scar tissue, we then excise scar tissue in the usual manner. At this point, reference and pacing electrodes are inserted on the right and left ventricular myocardia, respectively, while the patient is warmed to 38°C. Ventricular tachycardia is induced during total cardiopulmonary bypass by programmed ventricular stimulation, and epicardial and endocardial mapping is carried out, with particular attention given to the borders of the myocardial resection and to areas of interest located preoperatively. Resection of the arrhythmogenic focus is then done, the ventricle closed, air evacuated from the

heart and cardiopulmonary bypass discontinued.

During the past six months, we have used this technique in treating seven patients. All patients underwent coronary artery saphenous vein bypass grafts. Five of the seven had concomitant aneurysmectomy or excision of scar tissue in addition to subendocardial resection. In two patients, no resection was carried out because the tachycardia could not be induced after revascularization. One death occurred suddenly and late (presumably from an arrhythmia); ventricular tachycardia could not be stimulated in this patient intraoperatively.

Our research protocol has involved restudy of all patients six to eight weeks after their surgical operation. These studies include continuous electrocardiographic monitoring for at least 24 hours, exercise stress testing and repeat ventricular stimulation studies (Figure 11). To date, three patients are asymptomatic without antiarrhythmic drug therapy, three are asymptomatic while receiving standard antiarrhythmic drug therapy, and two of the three have not undergone repeat testing so that long-term need for therapy is not known. Although the reported series is small, we believe that techniques of cardiac mapping and resection of arrhythmogenic foci coupled with bypass grafting may offer improved results over coronary artery bypass operation and standard aneurysmectomy alone.

In summary, recent advances in our understanding of basic mechanisms of tachyarrhythmias have enabled development of newer approaches in the management of patients with tachyarrhythmias. In most patients, tachycardia can be controlled by appropriate drug therapy; promising experimental drugs such as the calcium blockers appear to effect excellent results in patients with supraventricular tachycardia. In patients who prove to be resistant to drug therapy, consideration should be given to the use of cardiac pacing or cardiac electrosurgical procedures in which His bundle or accessory pathway ablation or excision of ventricular arrhythmogenic foci may be carried out as described.

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Causes of Gastritis

GASTRITIS is a condition which until recently has not been truly appreciated in terms of its depth . . . both in the incidence and the degree of morbidity it can produce in the patient population. But, since we began using the flexible gastro-scope, it is recognized much more frequently; . . . it is not an increase of incidence . . . just an increased recognition rate. Some factors that play a role in acute gastritis are stress, infection and alcohol, aspirin, phenylbutazone (Butazolidin) and other chemicals . . . and most cases of acute gastritis result from chemical irritation to the mucosa.

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